Amendments to the Specification

Please replace paragraph **[0034]**, which appears in the specification at page 10, lines 3-15, with the following re-written paragraph. This paragraph was numbered [0044] in the published version ((US 2002/0146698 A1) of the application.

-- Among enzymes that catalyze conversion of a toxic oxygen species to a less toxic oxygen species, four are of particular relevance, namely mitochondrial manganese superoxide dismutase (MnSOD), cytoplasmic copper/zinc superoxide dismutase (CZSOD), catalase (CAT), and glutathione peroxidase (GP). Polymorphisms that occur in these genes are known to be associated with various disorders (see, e.g., Kimura et al., 2000, Am. J. Ophthalmol. 130:769-773). Occurrence of disorder-associated polymorphisms in at least one (and preferably two, three, or all) of these four genes should be assessed in the methods described herein, given the importance of these genes. Similarly, the kits described herein preferably include reagents for detecting ~~di~~-disorder-associated polymorphisms in at least one (and preferably two, three, or all) of these four genes. In addition, the significance of occurrence of disorder-associated polymorphisms in these genes can be applied by assigning a greater weighting factor to disorder-associated polymorphisms of these genes than to disorder-associated polymorphisms in other genes associated with oxidative stress. --

B1

Please replace paragraph **[0037]**, which appears in the specification at page 10, line 27, through page 11, line 5, with the following re-written paragraph. This paragraph was numbered [0047] in the published version ((US 2002/0146698 A1) of the application.

B2

-- Among the genes which exacerbate oxidative damage are genes which encode a protein that induces production of a toxic oxygen species, either directly (e.g., by catalyzing a reaction in which a toxic species of oxygen is a direct or side product) or indirectly (e.g., by enhancing flux through a metabolic pathway that leads to production of a toxic species of oxygen). Examples of proteins that directly or indirectly induce production of toxic oxygen species include myeloperoxidase, tumor necrosis factor alpha, NADH/NADPH oxidase p22 phox protein, nitric oxide synthase, xanthine oxidase, and cytochrome P450. Detection in a human genome of disorder-associated polymorphisms in one or more genes encoding one of these proteins indicates that the human exhibits enhanced susceptibility to oxidative damage. --